

## CASE REPORT

# Paraprosthetic leak unmasked by thrombolysis for thrombosed mitral valve

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Prosthetic valve thrombosis (PVT) is classically a cardiothoracic surgical emergency. Case series, however, report thrombolysis as first line management for PVT. A case of mitral PVT treated successfully with thrombolysis is described. Immediately after thrombolysis a trivial paraprosthetic leak noted on pretreatment transoesophageal echocardiography had increased significantly in severity. The paraprosthetic leak subsequently required repeat mitral valve replacement. It is speculated that the thrombolytic treatment interfered with the usual healing process by disrupting the fibrin deposited at the valve ring margin. This suggests that fibrin is important in the formation of the annular seal of the prosthetic valve and that patients receiving thrombolysis should be monitored for this complication.

Prosthetic valve thrombosis (PVT) is an uncommon but potentially fatal complication of prosthetic valve replacement. In a large study involving 988 patients with implanted St Jude mechanical prostheses at one centre over a 10 year period, the incidence of PVT was 1.7%.<sup>1</sup> Optimal management remains controversial and lies between thrombolysis, valve debridement, and repeat valve replacement. Thrombolysis has been recommended for high risk surgical candidates.<sup>2</sup> There are observational but no randomised data to guide management. Case series report clinical success for thrombolytic treatment for PVT in 70% of cases, but thrombolysis may be complicated by haemorrhage, systemic embolism in up to 18% of patients,<sup>3</sup> rethrombosis in 20%, and death in 10%.<sup>4</sup> We report a case of mitral PVT treated by thrombolysis. The thrombolysis successfully treated the valve thrombosis but immediately after thrombolysis a trivial paraprosthetic leak noted on pretreatment transoesophageal echocardiography (TOE) had increased significantly in severity. We speculate that the thrombolytic treatment may have interfered with the usual healing process by disrupting the fibrin deposited at the valve ring margin.

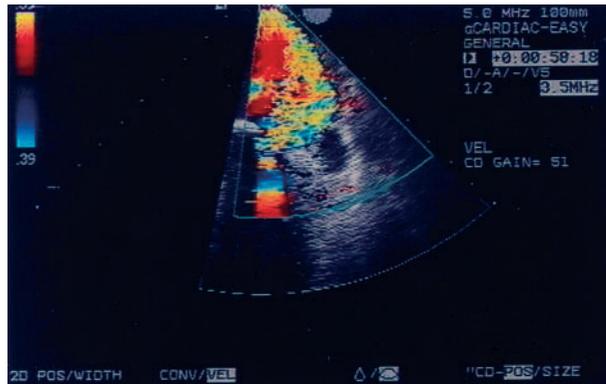
### CASE REPORT

A 73 year old man underwent mitral valve replacement with a 29 mm CarboMedics bileaflet prosthesis for symptomatic mitral regurgitation. Preoperative cardiac catheterisation showed moderate impairment of left ventricular systolic function with no significant coronary artery disease. He had a history of stroke without residual deficit and of permanent pacemaker insertion for atrial fibrillation with pauses. The initial postoperative hospital stay was uncomplicated and he was discharged six days later and prescribed warfarin.

Four days after discharge he suffered a transient ischaemic attack characterised by expressive dysphasia. Two weeks later he was readmitted to hospital with New York Heart Association functional class IV heart failure. Examination showed a diminished first heart sound and signs of



**Figure 1** Transoesophageal echocardiogram (TOE) showing spontaneous echo contrast within a dilated left atrium and greatly reduced excursion of both mitral prosthesis leaflets.



**Figure 2** Further TOE showing that severe paraprosthetic leak, with a broad based jet directed laterally and posteriorly extending around the back of the left atrium.

pulmonary oedema. The international normalised ratio was 1.7. Transthoracic echocardiography showed increased forward velocities across the mitral prosthesis with a peak velocity of 2 m/s (expected 1.4 m/s). The pressure half time was 345–405 ms (expected 80 ms) and the estimated valve area was 0.5–0.6 cm<sup>2</sup>. TOE showed spontaneous echo contrast within a dilated left atrium and greatly reduced excursion of both mitral prosthesis leaflets in keeping with valve thrombosis (fig 1). A trivial paraprosthetic leak at the lateral left atrium wall was noted.

**Abbreviations:** PVT, prosthetic valve thrombosis; TOE, transoesophageal echocardiography

After the management options had been discussed by the surgeon, cardiologist, and patient, tissue plasminogen activator was administered intravenously (15 mg bolus, 50 mg over 30 minutes, and 35 mg over 60 minutes). More aggressive anticoagulation was undertaken with warfarin and intravenous heparin was administered until the international normalised ratio was  $> 3.0$ . The following day the mechanical heart sounds were of noticeably higher pitch. Repeat TOE showed normal prosthetic leaflet motion. The peak forward velocity across the prosthesis had decreased to 1.5 m/s. The pressure half time was also much lower at 115 ms with an estimated valve area of 1.9 cm<sup>2</sup>. The trivial paraprosthetic leak noted on pretreatment TOE had, however, increased significantly in severity. Three weeks later, further TOE showed that the paraprosthetic leak was now severe with a broad based jet directed laterally and posteriorly extending around the back of the left atrium (fig 2). There was no clinical or bacteriological evidence of endocarditis and serum inflammatory markers were not increased. The mitral valve prosthesis was replaced with a 33 mm CarboMedics prosthesis. At surgery it was noted that the valve had not had a structural failure and that the sutures were intact, but there was no fibrous tissue over the sutures. The patient's postoperative recovery was uncomplicated.

## DISCUSSION

We have described a case of mitral PVT treated by thrombolysis. The thrombolytic treatment resulted in resolution of the valve obstruction, but this was accompanied by a significant increase in the severity of a previously trivial paraprosthetic leak. We speculate that the thrombolytic treatment may have been directly implicated in the increased severity of the paravalvar regurgitation. This hypothesis is supported by TOE evidence that paravalvar regurgitation increased within one day of thrombolytic treatment. Furthermore, there is a feasible mechanism through which

thrombolytic treatment may interfere with the usual healing process by disrupting fibrin deposited at the valve ring margin. This potentially interfered with the focus for cell migration and fibroblast deposition and proliferation with reduced formation of fibrous tissue.

This is the first report of thrombolytic treatment exacerbating a paraprosthetic leak. We conclude that the presence of even a trivial paraprosthetic leak should be one of the factors considered when determining the management of patients with PVT early after valve replacement. Patients with PVT treated with thrombolysis should be monitored for the development of paravalvar regurgitation.

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